Dear Sir,

Even if we appreciate Dr. Fisher’s remarks, we still do not feel that our approach has been inadequate. Our opinion is based on some evidence, although we could not perform any laboratory investigation to better define the type of heparin-associated thrombocytopenia (HAT) [1]. First, as is clearly demonstrated in figure 1 of our paper, the platelet count started to decrease on the day following the administration of heparin [2]. In our patient, we could exclude any previous exposure to heparin, which, according to the current literature, represents a prerequisite for the occurrence of HAT II immediately following the initiation of heparin therapy. In the absence of such an exposure, HAT II is reported to occur after some days of heparin treatment [3]. Second, the extremely low platelet count was probably related to their value which was already abnormal before the administration of heparin. Third, we could not discontinue heparin due to the ongoing continuous venovenous hemofiltration which required the administration of at least minimal amounts of heparin, as we did not have any safe alternative to its use, heparinoids not yet being available in our country at the time that we treated this patient. Moreover, we could not switch to prostacyclin due to the unstable hemodynamics. Anyway, being well aware of the possible complications related to the administration of heparin in these circumstances, we performed a number of investigations aimed to their early identification [2]. Indeed, as far as the treatment of both forms of HAT is concerned, we agree that a therapeutic option feasible in all patients is still lacking. However, we guess that in patients undergoing continuous venovenous hemofiltration and related techniques, the administration of a low-molecular weight heparin could be a reasonable option as a recent comparative study performed in a large number of patients failed to demonstrate any HAT after the administration [4].

References